Bacterial intracranial aneurysms

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 \checkmark The treatment of 13 patients with bacterial intracranial aneurysms is reported. The incidence of bacterial intracranial aneurysms was 4% of all patients admitted with intracranial aneurysms and 3% of all patients admitted with bacterial endocarditis. Each patient had neurological signs or symptoms suggestive of intracranial disease prior to the diagnosis of an aneurysm. Alpha *Streptococcus* was the most common infecting organism. All patients were treated with specific, high-dose antibiotics, and five patients underwent surgery as well. There were no surgical deaths. Six of eight nonsurgically treated patients died. A review of the literature confirms a high mortality for patients treated with only antibiotics, and a low mortality for elective surgery. The authors conclude that 1) patients with bacterial endocarditis, who develop sudden severe headache, focal neurological signs or symptoms, or seizures, should undergo serial cerebral angiography every 7 to 10 days throughout their hospitalization; 2) if an aneurysm is identified it should be excised whenever possible; and 3) patients with proximal or multiple aneurysms should be considered for surgery.

KEY WORDS · bacterial aneurysm · subarachnoid hemorrhage · mycotic aneurysm · bacterial endocarditis · cerebral aneurysm · serial angiography

ACTERIAL intracranial aneurysms are uncommon, but life-threatening, lesions. Controversy has recently arisen over the appropriate management of these lesions and has motivated this review. This paper reports the experience at a single hospital with bacterial intracranial aneurysms, and includes 13 patients. Drawing from this experience and the literature, we propose to examine the following questions: 1) Are there warning signs or symptoms that suggest the diagnosis of bacterial intracranial aneurysm prior to a hemorrhage? 2) Which patients with bacterial endocarditis should have cerebral angiography? 3) What is the role of serial angiography in following patients with bacterial endocarditis? 4) If an aneurysm has been documented, is medical therapy alone adequate? 5) Is there a recommended surgical management, and is it modified by the presence of proximal or multiple aneurysms?

The records were reviewed for all patients with a diagnosis of bacterial or mycotic intracranial aneurysm seen at the UCLA Center for the Health Sciences from June, 1955, to April, 1978. The diagnosis of bacterial aneurysm was established by pathological examination of surgical and autopsy specimens or by the finding of an aneurysm in the cerebral circulation of patients whose history, physical

examination, and blood cultures led to a diagnosis of bacterial endocarditis.

Illustrative Case Reports

Case 1

This 20-year-old white woman was known to have congenital subaortic stenosis. The patient was well until March 31, 1978, when she had an attack of bronchitis. Treatment consisted of oral ampicillin. Over the next 12 days, she developed neck stiffness, nonpruritic, erythematous, raised lesions on her legs, swollen, sore fingers, splinter hemorrhages, and fever elevations to 40°C. The patient was admitted to UCLA when she was found to be difficult to arouse.

Examination revealed a temperature of 37.4°C, hemorrhagic lesions on fingers, toes, palms, and soles, a small hemorrhage in the right retina, and a Grade III/VI aortic murmur. The patient was lethargic and had a nonfluent aphasia. The eyes were deviated to the left, the right side was paretic, and the neck was slightly stiff. The admitting diagnosis was bacterial endocarditis with a left frontal hemorrhage. The patient was started on intravenous penicillin, oxacillin, and gentamicin. All of 12 blood cultures grew *Staphylococcus aureus*. Computerized tomography (CT) on the day of admission showed a left frontoparietal hemorrhage, and arteriography showed an embolic filling defects at the parieto-occipital branch of the posterior cerebral artery and at the bifurcation of the pericallosal artery, and narrowing of the left opercular branches (Fig. 1 *left*).

Two weeks after admission, the patient had a generalized seizure and became unresponsive, with bilateral decerebrate posturing. A CT scan showed hemorrhage into the left temporal lobe and basal ganglia. Angiography showed aneurysms of the distal, left middle cerebral, left posterior cerebral, and the pericallosal arteries (Fig. 1 right). The hematoma was evacuated, and what was thought to be an aneurysm was clipped.

Angiography 2 weeks after operation showed enlargement of the left middle cerebral aneurysm to twice the original size, with no change in the other aneurysms. The enlarging aneurysm was excised 3 days later. Angiography was repeated 10 days after the second surgery and showed no middle cerebral artery aneurysm, no change in the remaining two aneurysms, and no further emboli. The patient was treated with antibiotics for 6 weeks. Her neurological examination at discharge showed a moderate nonfluent aphasia, and a right hemiparesis. Five months later she had a mild nonfluent aphasia and Grade 4 strength on the right. Soon after this examination the patient developed refractory heart failure and died.

Case 2

This 47-year-old white man had a history of a heart murmur but was previously in excellent health. He was admitted to UCLA Center for the Health Sciences three times in 1 month complaining of a persistent fever. On his third admission the physical examination was normal except for a temperature of 39.4°C, mental confusion, and a moderate nonfluent aphasia. Lumbar puncture showed an opening pressure of 220 mm H₂O, 729 white blood cells/cu cm (75% polymorphonuclear cells), 220 red blood cells/cu cm, a protein of 92 mg%, and a glucose of 58 mg% with a blood glucose of 122 mg%. Gram and acid-fast stains, india ink preparation, and cultures were all negative. The patient was admitted with a diagnosis of either meningitis or cerebral abscess, and was started on intravenous penicillin, intramuscular streptomycin, and triple antituberculous therapy. The day following admission, a Grade II/VI systolic murmur and an Osler's node on the ball of the left foot were noted. Bacterial endocarditis was diagnosed, and a cerebral angiogram was recommended to rule out a bacterial aneurysm.



FIG. 1. Case 1. Left internal carotid artery angiograms. Left: On admission, embolic filling defects are seen at the bifurcation of the pericallosal artery and the parieto-occipital branch of the posterior cerebral artery (double arrows). An anterior operculum branch is narrowed (single arrow). Right: Eleven days later, aneurysms are revealed at the locations of the previously demonstrated emboli or narrowing.

Case No.	Age (yrs), Sex	SAH	Underlying Heart Disease	Possible Predisposing Factors	Period BE to SAH† (days)
1	20, F	yes	subaortic stenosis	bronchitis	12
2‡	47, M	yes	mitral regurgitation		0
3‡	32, F	no		prolonged labor	unruptured
4	29, F	no		teeth cleaned	unruptured
5	25, M	yes	mitral regurgitation	teeth extraction	8
6	70, M	yes		urological surgery	23
7‡	52, M	yes			0
8‡	28, M	yes	mitral regurgitation		35
9	58, M	yes	00	dental work	35
10	39, F	ves		teeth cleaned,	5
		2		IV drugs used	
11	37, F	yes	mitral regurgitation	carious teeth	28
12	29. F	ves	2 8	teeth cleaned	24
13	52, F	yes		—	6

TABLE 1Patient population*

*SAH = subarachnoid hemorrhage; BE = bacterial endocarditis; IV = intravenous.

 † The number of days from the diagnosis of bacterial endocarditis to the diagnosis of subarachnoid hemorrhage.

‡Cases previously reported by Takahashi, et al.42

Angiography confirmed a left ascending parietal branch aneurysm (Fig. 2), which was excised 2 days later. The pathological diagnosis was a bacterial cerebral aneurysm. Postoperatively, the patient was neurologically normal. Angiography was repeated 3¹/₂ weeks after operation, and showed no new aneurysm and no filling of the previous aneurysm. The patient was discharged in good health after 6 weeks of antibiotic therapy.



FIG. 2. Case 2. Preoperative left internal carotid artery injection showing an ascending parietal branch aneurysm (arrows).

Summary of Cases

Patient Population

During the period of this study, there were 459 patients admitted with bacterial endocarditis and 296 patients with cerebral aneurysms. Thirteen patients fulfilled the requirements for bacterial intracranial aneurysm. Seven were female and six were male. Their ages ranged from 20 to 70 years, with an average age of 40 years (Table 1).

Five of thirteen patients had a past history of heart disease. Nine patients had possible predisposing conditions. Examples included dental work, bronchitis, carious teeth, prolonged labor, and urological surgery (Table 1).

Clinical Presentation

The diagnosis of bacterial endocarditis was made in all patients within 2 days of admission. This diagnosis preceded that of bacterial intracranial aneurysm in all but one case. The time from diagnosis of bacterial endocarditis to subarachnoid hemorrhage (SAH) ranged from 0 to 35 days, with an average of 18 days. Two patients with an aneurysm had not suffered an SAH. The diagnosis of bacterial intracranial aneurysm was made following the sudden onset of coma in six patients, after the onset of focal neurological findings in six patients, and after the sudden onset of severe headache in one patient.

TABLE 2Bacteriological findings in 12 patients

Organism Cultured	No. of Cases
alpha hemolytic Streptococcus	5
gamma Streptococcus	3
Staphylococcus aureus	2
beta hemolytic Streptococcus	1
Peptostreptococcus	1

TABLE 3 Radiographic results

Radiographic Procedure	Total Cases	Positive Results
omputerized tomography	6	5
ingiography	12	11

TABLE 4Results of serial angiography in six patients

Result	No. of Cases
enlargement of aneurysm	2
omplete resolution of aneurysm	1
ineurysm absent after excision	2
new aneurysm at a new site	1

TABLE	5	

Location of 19 aneurysms*

Location of Aneurysms	No. of Aneurysms
middle cerebral artery	15
anterior cerebral artery	2
posterior cerebral artery	2

*All aneurysms were beyond the first division of each individual vessel. Three patients had multiple aneurysms.

TABLE 6					
Treatment	and outcome	in	13	patients	

	Outcome			Total	
Therapy	Normal Disabled		Dead	Cases	
ntibiotics alone	2	0	6	8	
ntibiotics & operation	2	3	0	5	

Bacteriological Studies

Twelve of 13 patients had positive cultures, 11 from blood and one from an autopsy specimen (Table 2). A single organism was found in all 12 instances. The remaining patient (Case 2), who had not recently been treated with antibiotics, failed to grow organisms in spite of multiple cultures for aerobes, anaerobes, and fungi. The diagnosis of bacterial intracranial aneurysm was established in this case by pathological examination of the excised aneurysm. The most common organism was alpha *Streptococcus*.

Computerized Tomography

Computerized tomographic scans were obtained in six patients (Table 3). One was normal, one was performed 1 month after a diagnostic angiogram, and four were completed on the day of angiography. The latter four each demonstrated hematomas. The CT scan was positive for a hematoma in one patient who had no angiographically demonstrated aneurysm. The aneurysm was discovered at operation in this patient.

Angiography

Twelve patients underwent cerebral angiography, and at least one clearly defined aneurysm was seen in 11 (Table 3). A total of 19 aneurysms were found. The diagnosis of bacterial aneurysm was made at operation or autopsy in the two patients without angiographic evidence of an aneurysm. Serial angiograms were obtained in six patients. An aneurysm clearly enlarged in two cases, resolved in one case, and a new aneurysm appeared in one case (Table 4). There were three patients with multiple aneurysms. The locations included 15 middle cerebral, two anterior cerebral, and two on the posterior cerebral arteries (Table 5). All aneurysms were beyond the first major bifurcation of the involved vessel and were thus labelled as "distal."

Treatment

Twelve of 13 patients received a 6-week course of appropriate, high-dose, intravenous antibiotic therapy, beginning with the day of admission (Table 6). The 13th patient's treatment was started 2 days after admission, when the results of blood cultures were obtained. Craniotomy was performed in five patients. Eight patients received only antibiotic therapy. No patient received antihypertensive or antifibrinolytic therapy. Six of eight patients treated only with antibiotics died as a direct result of aneurysm rupture. The remaining two patients were neurologically normal at discharge. Of the five patients treated surgically, four were operated on after an SAH. Surgical treatment consisted of complete excision of four aneurysms and muscle-wrapping of one. There were no operative deaths and, at discharge, two patients were neurologically normal, one had a very mild monoparesis, and two remained hemiplegic as a result of their original hemorrhages.

Pathological Findings

Pathological analysis of the bacterial aneurysm was obtained in eight of 13 cases. All cases were described as showing enlarged or ruptured vessels with prominent inflammation and marked attenuation of the vessel wall with wall necrosis.

Discussion

Bohmfalk, et al.,⁵ summarized 85 cases of bacterial intracranial aneurysm reported between 1954 and 1978. We have reviewed these cases from the literature^{1-7,9-14,16-23,26-29,31-38,40-44} and have compared the results with our own.

Patient Population

The age, sex, incidence of bacterial intracranial aneurysm in all patients with intracranial aneurysm, and the incidence of bacterial intracranial aneurysm in patients with bacterial endocarditis in our series were similar to others.^{4-6,8,15,17,36,87,39} The incidence in our patients of underlying heart disease, predisposing conditions, and the spectrum of these conditions was also similar to the literature.^{8,35}

Pathogenesis

In 1968, Nakata, *et al.*,³⁰ published experimental results showing that after the introduction of bacteria into the dog aorta, which had been isolated by temporary clamps just above the renal arteries and the aortic bifurcation, vascular wall destruction began in the layers adjacent to the vasa vasorum. They concluded that stasis and sepsis in the vasa vasorum were the necessary initiating events for the formation of a bacterial aneurysm.

Molinari, et al.,24,25 pointed out that, although the original site of infection might be the vasa vasorum, this was rarely present in the cerebral vessels, particularly in distal branches where bacterial aneurysms were most likely to occur. From their own experimental model of bacterial emboli introduced into the cerebral circulation, they were able to conclude that aneurysms consistently appeared at the site of an infected embolus and that the earliest changes in the vessel wall occurred in the adventitial layer and spread to involve the media. They proposed that "in the absence of the vasa vasorum, bacteria could escape through occluded origins of the thin-walled penetrating vessels to the Virchow-Robin space and then to the adventitia of the parent vessel."25 They explained that the aneurysmal enlargement was produced by pulsations against the necrotic wall of an occluded vessel or by those same pulsations against a weakened wall of a recanalized vessel. This latter process might explain the late appearance of an aneurysm during an-

TABLE 7Signs and symptoms prior to hemorrhage

Symptoms or Signs	No. of Cases	
 sudden severe headache	6	
focal deficits		
aphasia	3	
hemiparesis	4	
facial weakness	1	
facial numbness	1	
seizures	1	

tibiotic treatment, as occurred in Case 1. Molinari²⁴ stressed the short time period (1 to 3 days) between lodging of an infected embolus in a cerebral vessel and aneurysm formation in dogs not treated with antibiotics, or infected with virulent organisms. However, Molinari and his co-workers²⁵ also noted that, if dogs were partially treated with an appropriate antibiotic, aneurysm rupture no longer regularly occurred in 1 to 3 days, but aneurysms were present when the dogs were sacrificed 7 or 8 days after embolization. These results would suggest that an aneurysm may develop in a patient with bacterial endocarditis who is appropriately treated, in as short a time as 1 week.

Clinical Presentation and Angiography

The neurological signs and symptoms of patients who develop bacterial intracranial aneurysms are variable,¹⁷ but are often secondary to one of three processes: 1) septic embolization; 2) meningitis; and 3) SAH. The first two produce signs and symptoms before aneurysm formation, and are infectious processes that may go on to aneurysm formation, as noted by Molinari, *et al.*,^{24,25} and Suwanwela, *et al.*⁴⁰ The third is a direct result of aneurysm rupture, and is frequently associated with disastrous results. Most authors have expressed concern that there are no early warning signs to herald aneurysm formation except SAH, and, thus, unless all patients with bacterial endocarditis undergo serial angiograms, the mortality rate cannot be reduced.

A review of our patients suggests otherwise. Each of our cases was accompanied by premonitory signs or symptoms suggestive of intracranial disease (Table 7). In six patients who survived, these events initiated angiography. Case 2 is an example. One other patient who survived had early signs and symptoms, but angiography was not done until she suddenly became hemiplegic. The six patients who died as a direct result of an intracranial hemorrhage also had warning signs or symptoms. In two patients (Table 8), these symptoms occurred only a few hours before a catastrophic

Case No.	Signs or Symptoms	Delay* (days)	Discharge Status	
 1	hemiparesis, decreased mental status	12	disabled	
2	nonfluent aphasia, decreased mental status	13	normal	
3	sudden severe headache, hemiparesis	13	disabled	
4	sudden severe headache	5	normal	
5	sudden severe headache	1/8	dead	
6	seizures, decreased mental status	4	dead	
7	sudden severe headache	1/4	dead	
8	facial weakness, personality change	6	dead	
9	hemiparesis	35	dead	
10	sudden severe headache	1	normal	
[]	nonfluent aphasia, personality change	1	normal	
12	sudden severe headache	29	dead	
13	nonfluent aphasia	8	disabled	

 TABLE 8

 Relationship of signs and symptoms to the diagnosis of an aneurysm

*Number of days from onset of warning signs or symptoms to catastrophic hemorrhage or angiographic diagnosis of an aneurysm.

event. It is doubtful whether early diagnostic procedures would have changed their clinical course. However, the remaining four patients had signs or symptoms days before a catastrophic event, which, if correctly interpreted, could have led to early angiography and definitive treatment. Our results suggest that those patients who develop a sudden severe headache, focal neurological deficits, or seizures, should be selected for angiography. It is recognized that 28% to 39% of patients with bacterial endocarditis may present with neurological signs or symptoms.^{17,35} However, only 19% have been reported with the above signs or symptoms,¹⁷ and one could expect that at least 25% to 33% of these patients (4% to 6% of all bacterial endocarditis patients) would have a cerebral bacterial aneurysm.

Serial Angiography

Several points should be noted from our cases and the literature: 1) rupture of a bacterial intracranial aneurysm may occur at any point in the course of appropriate antibiotic therapy; 2) a new aneurysm may appear in subsequent angiograms even though previous angiograms were negative; and 3) new aneurysms may appear after excision of a solitary aneurysm.^{4,5,17-19,23,37,44} These facts emphasize the need for serial angiography throughout hospitalization. The experimental literature indicated that aneurysms are present 7 days after a septic embolus to the brain.25 Our own experience demonstrates that there is an average of 10 days from the onset of warning signs or symptoms to a catastrophic hemorrhage or to the angiographic demonstration of a cerebral aneurysm (Table 8). Therefore, three-vessel cerebral angiography at the onset of these signs or symptoms and repeated every 7 to 10 days during 6 weeks of antibiotic therapy, even after surgical excision of one aneurysm, would seem to be the method and frequency that would be most reliable for detecting bacterial cerebral aneurysms.

Computerized tomography has not been helpful in establishing a diagnosis of bacterial intracranial aneurysm, and it seems unlikely that in the near future it can do little more than establish the presence of a hemorrhage, abscess, or infarct in these patients. Importantly, a 6-week course of antibiotic therapy does not insure the prevention of further aneurysm formation,² but, as demonstrated by our data (Table 1), most episodes of SAH occurred within 35 days of the diagnosis of bacterial endocarditis, and thus before completion of antibiotic therapy.

Treatment: Surgical or Nonsurgical

Bingham,⁴ in a review of 45 patients with bacterial intracranial aneurysms, emphasized that 11 of 21 patients followed with serial angiography had complete resolution of their aneurysms, and an additional six patients of this group had a decrease in aneurysm size. This led him to suggest serial angiography for all patients with a documented aneurysm. He recommended an operation only if the aneurysm enlarged, or did not change in size after 6 weeks of antibiotic therapy. He reported that three of 20 patients, who received only antibiotic therapy, died, and six of 25 patients, who received combined antibiotic and surgical treatment, died. He noted that the higher mortality rate associated with operation was not a true reflection of surgical mortality, which he estimated to be quite low.

Bohmfalk, et al.,⁵ reviewed 85 patients, including the patients in Bingham's series. They noted that an aneurysm resolved completely in 10 of 25 patients treated with antibiotics alone, and followed with serial angiography. This group of patients, as with

Bacterial intracranial aneurysms

TABLE 9 Bacterial intracranial aneurysms: treatment and mortality in three series

Authors	Antibiotics Only		Antibiotics & Surgery	
& Year	Total Cases	Dead	Total Cases	Dead
Bingham, 1977	20	3	25	6
Bohmfalk, et al., 1978	38	20	30	8
Frazee, et al., 1980	8	6	5	0

 TABLE 10

 Treatment and mortality in previously reported cases*

Treatment	Total Cases	Dead	
proximal aneurysms*		1	
nonsurgical	3 9	6	
multiple aneurysms			
operation nonsurgical	4 11	0† 0‡	

*Treatment and outcome is unknown for one patient. †No surgically treated patient died as a result of operation. ‡The results of treatment for two patients are unknown.

Bingham's series, was a selected population and excluded the majority of patients (60 of 85 and 24 of 45, respectively) who were not followed by serial angiography. We emphasize that, although bacterial cerebral aneurysms resolve or decrease in size in some patients, the mortality of antibiotic therapy alone is high. Bohmfalk and colleagues reported that 20 of 38 patients in this group died. There were, however, no deaths for elective surgical procedures. Eight of 13 patients who required emergency operations died, but a careful examination of these eight patients confirms Bingham's earlier opinion that in most instances the deaths were not a result of surgery, but secondary to the already moribund state of the patient, rupture of a second aneurysm after the first had been successfully treated, or from unrelated causes. Our results underscore the high mortality associated with conservative therapy alone (six of eight patients) and the low mortality for craniotomy plus antibiotics (0 of five patients) (Table 9). These data suggest that therapy should consist of excision of the aneurysm as well as antibiotic treatment whenever possible.

Proximal and Multiple Aneurysms

The most recent comprehensive review⁵ suggests nonsurgical treatment for proximal and multiple aneurysms unless the latter show enlargement. A review of the treatment and outcome of the 13 cases of proximal aneurysm reported in the literature,^{2,4,6,18,28,33,36,87,41,44} excluding cavernous sinus aneurysms, shows one surgical death in three patients with operation; six of 10 patients died when treated with antibiotics alone (Table 10). The outcome is unknown for one patient in this latter group. The surgical group is too small to allow us to draw conclusions, but the high mortality associated with nonoperative treatment (two-thirds of the cases) should lead one to carefully consider an operation in each case. The concern for cerebral infarction as a complication of excision of a proximal aneurysm is obvious, but several alternative approaches should be examined before relying upon antibiotic treatment alone. These would include clipping of the aneurysm, excision with a superficial temporal artery to middle cerebral artery bypass, trapping of the aneurysm with or without a bypass, and wrapping of the aneurysm with muscle. We have had no experience with bypass surgery in these cases.

Interestingly, two of the previously reported "proximal" aneurysms were described as "berry" aneurysms, not bacterial,³⁶ while a third occurred approximately 2 years after endocarditis.² Thus, these aneurysms may have been preexisting saccular aneurysms that became secondarily infected.

Finally, we must consider the appropriate therapy for multiple aneurysms. A review of 15 such cases reported previously^{3-5,7,10,13,14,17,23,34,37,40} shows that four patients died, and 11 recovered. Four patients were treated surgically and 11 nonsurgically (Table 10). No patient died as a direct result of treatment. It is unclear why the mortality was unusually low in the nonsurgically treated group. However, as noted before, mortality for nonsurgically treated patients in general is approximately 50%, and this fact should be kept in mind when selecting the most appropriate therapy.

We believe that most patients with multiple aneurysms are surgical candidates. If multiple aneurysms are unilateral, they should be excised at one operation whenever possible. If they are bilateral, the largest aneurysm or the one presumed to have bled should be excised first. An appropriate recovery period should be allowed and angiography repeated before excision of the remaining aneurysm.

Conclusions

In view of the serious consequences of a ruptured bacterial intracranial aneurysm, the high morbidity of nonsurgical therapy, the low elective surgical mortality, and the rapid appearance of new aneurysms in some patients, we would recommend aggressive management for patients with bacterial endocarditis. Specifically, we suggest that 1) those patients with the diagnosis of bacterial endocarditis, who develop sudden severe headache, focal neurological signs or symptoms, or seizures, should undergo serial cerebral angiography every 7 to 10 days throughout their hospitalization; 2) if an aneurysm is identified, it should be excised whenever possible; and 3) patients with proximal or multiple aneurysms should be considered for operation, but that therapy must be individualized.

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References

- Agnoli A, Bettag W: Endokarditis und Subarachnoidalblutung. Z Neurol 199:295-305, 1971
- Alajouanine T, Castaigne P, Lhermitte F, et al: Cerebral arteritis of bacterial endocarditis: its late complications. JAMA 170:1858, 1959 (Abstract)
- 3. Bell WE, Butler C 2nd: Cerebral mycotic aneurysms in children. Two case reports. Neurology 18:81-86, 1968
- Bingham WF: Treatment of mycotic intracranial aneurysms. J Neurosurg 46:428-437, 1977
- 5. Bohmfalk GL, Story JL, Wissinger JP, et al: Bacterial intracranial aneurysm. J Neurosurg 48:369-382, 1978
- Cantu RC, LeMay M, Wilkinson HA: The importance of repeated angiography in the treatment of mycoticembolic intracranial aneurysms. J Neurosurg 25: 189-193, 1966
- 7. Castaigne P, Laplane D, Ricou P, et al: [Multiple intracranial arterial aneurysm of mycotic appearance. Repeated vascular embolic accidents. Myxoma of the left ventricle]. **Rev Neurol 131:**339-346, 1975 (Fr)
- Cates JE, Christie RV: Subacute bacterial endocarditis. Q J Med 20:93-130, 1951
- Devadiga KV, Mathai KV, Chandy J: Spontaneous cure of intracavernous aneurysm of the internal carotid artery in a 14-month-old child. Case report. J Neurosurg 30:165-168, 1969
- Gilroy J, Andaya L, Thomas VJ: Intracranial mycotic aneurysms and subacute bacterial endocarditis in heroin addiction. Neurology 23:1193-1198, 1973
- Hannesson B, Sachs E Jr: Mycotic aneurysms following purulent meningitis. Report of a case with recovery and review of the literature. Acta Neurochir 24:305-313, 1971
- Harrison MJG, Hampton JR: Neurological presentation of bacterial endocarditis. Br Med J 2:148-151, 1967
- Heidelberger KP, Layton WM Jr, Fisher RG: Multiple cerebral mycotic aneurysms complicating post-traumatic pseudomonas meningitis. Case report. J Neurosurg 29:631-635, 1968
- Hourihane JB: Ruptured mycotic intracranial aneurysm. A report of three cases. Vasc Surg 4:21-29, 1970
- 15. Housepian EM, Pool JL: A systematic analysis of intracranial aneurysms from the autopsy file of the Presbyterian Hospital, 1914 to 1956. J Neuropathol Exp Neurol 17:409-425, 1958
- Ishikawa M, Waga S, Moritake K, et al: Cerebral bacterial aneurysms: report of three cases. Surg Neurol 2:257-261, 1974
- Jones RH Jr, Siekert RG, Geraci JE: Neurologic manifestations of bacterial endocarditis. Ann Intern Med 71:21-28, 1969

- Katz RI, Goldberg HI, Selzer ME: Mycotic aneurysm. Case report with novel sequential angiographic findings. Arch Intern Med 134:939-942, 1974
- 19. King AB: Successful surgical treatment of an intracranial mycotic aneurysm complicated by a subdural hematoma. J Neurosurg 17:788-791, 1960
- Laguna J, Derby BM, Chase R: Cardiobacterium hominis endocarditis with cerebral mycotic aneurysm. Arch Neurol 32:638-639, 1975
- Lansky LL, Maxwell JA: Mycotic aneurysm of the internal carotid artery in an unusual intra-cranial location. Dev Med Child Neurol 17:79-88, 1975
- 22. Matson DD: Intracranial arterial aneurysms in childhood. J Neurosurg 23:578-583, 1965
- McNeel D, Evans RA, Ory EM: Angiography of cerebral mycotic aneurysms. Acta Radiol (Diagn) 9: 407-412, 1969
- 24. Molinari GF: Septic cerebral embolism. Stroke 3: 117-122, 1972
- Molinari GF, Smith L, Goldstein MN, et al: Pathogenesis of cerebral mycotic aneurysms. Neurology 23: 325-332, 1973
- Montrieul B, Terrasse J, Alix B, et al: [The relationship between intracerebral hematoma, mycotic aneurysm, *Staphylococcal* septicemia and endocarditis.] Sem Hop Paris 49:2009-2013, 1973 (Fr)
- Morgan WL, Bland EF: Bacterial endocarditis in the antibiotic era with special reference to the later complications. Circulation 19:753-765, 1959
- Morin MA, Talalla A: Angiography for mycotic aneurysm. N Engl J Med 281:1249-1250, 1969 (Letter)
- Moskowitz MA, Rosenbaum AE, Tyler HR: Angiographically monitored resolution of cerebral mycotic aneurysms. Neurology 24:1103–1108, 1974
- Nakata Y, Shionoya S, Kamiya K: Pathogenesis of mycotic aneurysm. Angiology 19:593-601, 1968
- Ng KK, Wong WK, Skene-Smith H: Ruptured mycotic intracranial aneurysm. Australas Radiol 19:255-257, 1975
- 32. Noonan JA, Wilson CB, Spencer FC, et al: Cerebral and cardiac complications from bacterial endocarditis. A successfully managed case with unusual complications. Am J Dis Child 116:666-674, 1968
- North-Coombes D, Schonland MM: Cerebral mycotic aneurysm. A case report. S Afr Med J 48:1808-1810, 1974
- Ojemann RG, New PFJ, Fleming TC: Intracranial aneurysms associated with bacterial meningitis. Neurology 16:1222-1226, 1966
- Pankey GA: Acute bacterial endocarditis at the University of Minnesota Hospitals, 1939–1959. Am Heart J 64:583-591, 1962
- Ray H, Wahal KM: Subarachnoid hemorrhage in subacute bacterial endocarditis. Neurology 7:265-269, 1957
- Roach MR, Drake CG: Ruptured cerebral aneurysms caused by micro-organisms. N Engl J Med 273: 240-244, 1965
- Shibuya S, Igarashi S, Amo T, et al: Mycotic aneurysms of the internal carotid artery. Case report. J Neurosurg 44:105-108, 1976
- Snow RM, Cobbs CG: Treatment of complications of infective endocarditis, in Kaye D (ed): Infective Endocarditis. Baltimore: University Park Press, 1976, pp 213-228
- 40. Suwanwela C, Suwanwela N, Charuchinda S, et al: In-

Bacterial intracranial aneurysms

tracranial mycotic aneurysms of extravascular origin. J Neurosurg 36:552-559, 1972

- 41. Sypert GW, Young HF: Ruptured mycotic pericallosal aneurysm with meningitis due to *Neisseria meningitidis* infection. Case report. J Neurosurg 37:467-469, 1972
- 42. Takahashi M, Killefer F, Wilson G: Cerebral mycotic aneurysms. Nippon Acta Radiol 30:489-499, 1970
- 43. Yarnell PR, Stears J: Intracerebral hemorrhage and occult sepsis. Neurology 24:870-873, 1974
- 44. Ziment I, Johnson BL Jr: Angiography in the management of intracranial mycotic aneurysms. Arch Intern Med 122:349-352, 1968

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